SUN-LB88

THYROTOXIC PERIODIC PARALYSIS IN HISPANIC PATIENTS BACKGROUND: Thyrotoxic periodic paralysis (TPP) presents as acute intermittent attacks of weakness related to hypokalemia, commonly reported in Asians and rare in Hispanics(1). Patients with TPP will have triiodothyronine (T3) triggered increased Na+/K+ ATPase pump activity and transcription of the KCNJ18 gene that encodes for the Kir2.6 channel(2). This permits insulin, catecholamines, stress and alcohol(3) to increase cellular intake of potassium, which causes depolarization and leads to weakness and paralysis. We report a case of TPP in a young Hispanic man who presented with lower extremity weakness and falls. **CASE PRESENTATION:** A 34-year-old Hispanic man with Graves' disease, nonadherent to medications presented with generalized weakness, more pronounced in legs, and recurrent falls. Physical examination was unremarkable except for mild enlargement of thyroid gland and abnormal gait due to weakness. Laboratory data showed hypokalemia of 1.8 mmol/L (3.7-5.1 mmol/L) and a TSH level of <0.004 mIU/L (0.34-5.6 mIU/L). Free T4 3.74 ng/dL (0.6-1.6 ng/dL), free T3 597 pg/dL (230-420 Pg/dL), thyroid stimulating Ig 148 (<130). Electrocardiogram did not show U waves. Radio iodine 123 scan of thyroid revealed diffusely increased 24-hour radioactive uptake of 66.5% (10-30%). The patient was diagnosed with TPP and supplemented with three doses of potassium 40 mEq IV infusion. Methimazole and metoprolol were started. He made a good clinical recovery within days. After discharge, he was treated with I-131 (13 mci) and developed postablative hypothyroidism on long term. He was euthyroid on levothyroxine. He did not have any recurrence of weakness at 7-year follow-up. **CONCLUSION:** TPP is uncommonly seen in Hispanics patients as opposed to Asians(3). Physicians should consider TPP as part of the differential diagnosis in young hyperthyroid Hispanic men presenting with weakness or paralysis, as early recognition and treatment can reduce recovery time and potentially prevent tachyarrhythmia or death. **REFERENCES:** 1. Matta A, Koppala J, Gossman W. Thyrotoxic hypokalaemic periodic paralysis: a rare presentation of Graves' disease in a Hispanic patient. BMJ Case Rep. 2014;2014. 2. Ryan DP, Ptacek LJ. Mutations in Potassium Channel Kir2.6 Cause Susceptibility to Thyrotoxic Hypokalemic Periodic Paralysis. Cell, 140(1), pp.88-98. 3. Amblee, A. and Gulati, S. (2016). Thyrotoxic Periodic Paralysis: Eight Cases in Males of Hispanic Origin from a Single Hospital. *AACE Clinical Case Reports*, 2(1), pp.e58-e64.

Steroid Hormones and Receptors

STEROID AND NUCLEAR RECEPTORS

Endocannabinoids Induce Endoplasmic Reticulum Stress in Hepatocytes and Human Coronary Artery Endothelial Cells

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SUN-LB137

Obesity and diabetes are important risk factors for the development of coronary heart disease and stroke. Plasma endocannabinoid (EC) levels are inappropriately elevated in obesity and diabetes, and are hypothesized to play a causal role in central regulation of weight gain. Importantly, it was recently demonstrated that cannabinoid receptor 1 (CNR1) triggers cell stress and induces apoptosis in kidney tubule cells exposed to palmitic acid and high-glucose (HG). HepG2 and human coronary artery endothelial cells (HCAEC) were treated with tunicamycin (TM), thapsigargin (TG), high-glucose (HG), anandamide (AN), and 2-arachondonyl glycerol (2-AG), and endoplasmic reticulum (ER) stress was measured. In cells treated with TM, AM, and 2-AG and the UPR inhibitors 4-phenylbutyrate (4-PB) and taurodeoxycholic acid (TUDCA), both 4-PB and TUDCA prevented AN and 2-AG from promoting ER stress. ER stress in cells treated with AN and 2-AG, but not TM, was inhibited by the CNR1 antagonist rimonabant. Similar results were obtained with HCAEC. Furthermore, treatment with AN and 2-AG induced inositol requiring enzyme 1α and protein kinase R-like endoplasmic reticulum kinase phosphorylation but had no effect on their expression, while activating transcription factor 6 and binding immunoglobulin protein expression were also induced by AN and 2-AG in both HepG2 and HCAEC. Finally, AN and 2-AG treatment induced CNR1 expression in both cell lines. These results strongly suggest that EC's promote ER stress and potentially induce liver and endothelial cell dysfunction.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Multicentric Carpotarsal Osteolysis Syndrome (MCTO) Has a Generalized High Turnover Bone Phenotype, High S RANKL and Responds to Denosumab

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MON-LB72

Background:MCTO is a rare disorder, caused by mutations in the MABF gene, a negative regulator of RANKL. Manifestations include carpal tarsal osteolysis and subsequent renal failure in some. Pathophysiology is poorly understood, and no effective treatment is available. **Clinical case:**A 5y old boy presented (2011) with R wrist pain and diffuse swelling. MRI showed pan-carpal synovitis with joint effusion. He did not respond to different anti-inflammatory medications. Plain films showed central